Skiing Associated Stroke: Causes, Treatment, and Outcome

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Background: Previous studies have described ischemic stroke temporally related to specific triggers, but only 1 series collected patients with acute ischemic stroke (AIS) following downhill skiing and all caused by cervical artery dissections. Here we describe our series of AIS temporally associated to ski practice, focusing on the frequency, pathogenesis, clinical presentation, and prognosis. *Methods:* We maintained a prospective list of Skiing Associated Strokes (SASs) from 2003 to 2017. From all AIS patients included in our stroke registry Acute Stroke Registry and Analysis of Lausanne (ASTRAL) over the same period, we identified a comparison group of non-SAS patients, matched for age and gender. *Results:* In the 12-year observation period, we identified 17 SASs (4 females, median age 51 years) and 51 matched control patients with nonski-associated strokes. Vascular risk factors, stroke features, and outcome were similar between the 2 groups. Stroke mechanism was arterial dissection in 11 of 17 SASs (65%) and in 7 of 51 control patients (14%, chi-square test: $P < .05$). In the other 6 cases of ski-associated stroke, etiology was cardiac embolism from atrial fibrillation in 2 patients, large vessel atherosclerosis with stenosis >50% in 1 patient, and undetermined in 3. Among the 11 patients with SAS caused by dissection, 8 reported minor falls while skiing, 1 had a major head trauma without loss of consciousness, and 2 had no traumatism (compared to preceding trauma in 29 of 147 [20%] of all other AIS caused by arterial dissection in ASTRAL, $P < .01$). *Conclusions:* Arterial dissection was a significantly more frequent stroke mechanism in SAS compared to matched controls, but other mechanisms occurred as well. Minor or moderate skiing-related trauma preceded most SAS with dissections.
Key Words: Epidemiology—stroke subtypes—risk factors—dissections

Introduction

While chronic risk factors for ischemic stroke are well identified, acute causative events or trigger situations are less recognized.[1] Previous studies have described ischemic stroke temporally related to specific situations, such as sport activity, yoga, cervical manipulation, or visits to the hairdressers and in the majority of patients, stroke was secondary to cervical artery dissections.[2,3] A single case series described patients with acute ischemic stroke (AIS) following skiing and all were caused by cervical artery dissections.[4]

In this study, we report a series of AIS temporally related to downhill ski practice Skiing Associated Strokes

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(SASs) focusing on frequency, pathogenesis, clinical presentation, and prognosis.

Methods

Here, we considered AIS "skiing-associated" if symptoms onset occurred during or shortly after skiing activity, or if the patient presented transient ischemic attacks or symptoms of arterial dissections causing the AIS while skiing. One of the authors (P.M.) maintained a prospective list of SASs from 2003 to February 27, 2017. Using a 1:3 exact age and sex matching of SASs and controls respectively, we randomly selected a control group of nonski-associated AIS patients from patients included in our hospital stroke registry "Acute Stroke Registry and Analysis of Lausanne" (ASTRAL)[5] for the same study period.

For all SAS and control group patients, we collected demographics, vascular risk factors, vascular territory, National Institute of Health Stroke Scale score, and revascularization treatment data. The stroke mechanisms were classified according to the Trial of Org 10172 in Acute Stroke Treatment criteria, with dissections considered as a separate mechanism. We reviewed acute neuroimaging for ischemic lesions and arterial pathology. The diagnosis of cervical artery dissection required visualization of intramural hematoma and narrowing/occlusion of the arterial lumen on multidetector high resolution Computed Tomography (CT) and CT angiography (CTA) (axial slices with section thickness = 1.25 mm before 2006 and .63 mm thereafter performed on a 16−detector row CT scanner−LightSpeed 16 Advantage; GE Healthcare, Milwaukee, Wisconsin until November 2005 and afterwards on a 64−detector row CT scanner−LightSpeed VCT 64; GE Healthcare) or Magnetic Resonance Imaging (MRI) and MRI angiography (among all 158 patient with dissection 77 had CT/CT angiography only, 81 had also MRI/MRI angiography). Outcome at 3 months was assessed in the outpatient stroke clinic or by telephone interview by mRS-certified medical staff. We considered a favorable outcome if the mRS was ≤2. For SAS patients, we collected additional information regarding occurrence and severity of fall and traumatism while skiing and on the delay between skiing and the first neurological symptoms.

We performed comparisons between SAS stroke patients and the control group with Fisher's exact test for categorical data and Kruskal-Wallis tests for numeric variables.

We carried out a second comparison between SAS caused by arterial dissection patients and all other patients with AIS caused by arterial dissection in ASTRAL during the study period (n = 147).

We performed statistical analysis with R statistical software (version 3.3.2, R Core Team [2016], R Foundation for Statistical Computing, Vienna, Austria). $P < .05$ were considered significant.

The ethics commission for research on humans of the Canton of Vaud approved collection, analysis, and publication of data from ASTRAL; for retrospective analysis of these data no informed consent is required according to the legislation of the Canton de Vaud, Switzerland.

Results

In the 12-year observation period, we identified 17 SASs (16 performing downhill skiing and 1 snowboarding, 4 females and 13 males, with median age of 51 years) and 51 non-SASs matched for age and gender (Table 1). Patient and stroke's characteristics were similar between the 2 groups, except for baseline National Institute of Health Stroke Scale, clearly higher in SAS patients (11 versus 4 points for controls), but not statistically significant. In both groups vascular risk factors were highly prevalent (at least 1 risk factor in >50% of patients) and we observed a high prevalence of smoking. Stroke mechanism was arterial dissection in 11 out of 17 ski-associated strokes (65%) and in 6 of the 51 control patients (12%, chi-square test: $P < .05$). In the other 6 cases of ski-associated stroke, etiology was cardiac embolism from atrial fibrillation in 2 patients, large vessel atherosclerosis with stenosis >50% in 1 patient and undetermined in 3. The majority of patients (82.4%) with SAS had favorable outcome and only 1 experienced stroke recurrence 1 year after the first event, with both index and recurrent event of cardioembolic origin.

In the 6 SASs without dissection, none had traumatism during skiing; all strokes happened in the day while skiing. None of the 6 patients reported symptoms preceding stroke.

Among the 11 patients with SAS caused by dissection, 6 reported symptoms preceding stroke onset (cervical pain, Claude-Bernard-Horner, or transient ischemic attack), all starting during or shortly after skiing. In 8 of the SAS patients with dissections, stroke onset followed ski practice by 1-30 days and in only 3 cases, stroke happened while skiing. Seven dissection patients reported minor falls while skiing (single or repeated), 1 reported a fall with neck hyperextension, 1 had a major head trauma without loss of consciousness, and 2 reported no fall or traumatism (Table 2): overall the frequency of preceding trauma in patients with SAS caused by dissection was 9 of 11 (82%), significantly higher than in all other AIS from arterial dissection in ASTRAL (29 of 147[20%], $P < .01$). Compared to all other dissections in ASTRAL (n = 147), skiing-associated dissections showed higher proportion of males (8 of 11 [73%], versus 80 of 147 [54%] in nonskiing-associated dissections) and smokers (6 of 11 [54%], versus 35 of 147 [24%] in nonskiing-associated dissections) and lower rate of significant stenosis (>50%) or complete arterial occlusion (**Table 3**). Three-month outcome measures were not different between the 2 groups: mRS ≤2 in 8 of 11(72%) versus 85 of 151(58%) for ski-associated dissections versus nonski-associated respectively, $P > .05$.

Discussion

We report a second series of SASs and carry out a systematic comparison of these patients with other matched-stroke patients and other patients with dissections.

SAS was frequently caused by arterial dissections, but one third was unrelated to dissections. Except for a higher initial stroke severity, SASs had characteristics similar to matched stroke patients. Both groups displayed a high prevalence of vascular risk factors with at least 1 risk factor in >50% of patients. Of note, smoking was highly prevalent in SAS caused by dissections, twice as high as in dissection nonski-associated, that featured a low prevalence of smoking in line with data in literature.[6] Observations from previous studies suggest that, once a dissection has occurred, smoking may increase the risk of stroke through its effects on endothelial function and thrombus formation.[7] Similarly, the high frequency of smoking in our SAS patients with dissection may suggest that smoking, prompting arterial wall inflammation, and fragility predisposes to dissection itself in case of minor traumatism as those observed in the majority of our SAS caused by dissection. Minor or moderate traumatism or falls while skiing were frequent in SAS patients with stroke related to arterial dissection and the frequency of

Table 1. *Comparison between ski-related and non ski-related stroke patients matched for age and gender*

	Strokes non ski-associated (n = 51)	Ski-associated stroke (n = 17)	*P* value
Age	50.9 (46.6-54.1)	51 (45.1-53.9)	.89
Female sex	12 (23.5%)	4 (23.5%)	1.00
mRS prestroke	0 (0-0)	0 (0-0)	.54
Previous stroke	4 (7.8%)	1 (5.9%)	1.00
Hypertension	20 (39.2%)	6 (35.3%)	1.00
Diabetes	9 (17.6%)	1 (5.9%)	.43
Hypercholesterolemia	32 (62.8%)	12 (70.6%)	.77
Smoking	18 (36%)	7 (41.2%)	.93
Atrial fibrillation	7 (13.7%)	2 (11.8%)	1.00
Coronary artery disease	6 (11.8%)	0 (0%)	.32
Patent forame ovale	10 (41.7%)	1 (12.5%)	.28
Body Mass Index	25 (23-27)	26 (23-27.3)	.85
Stroke arterial territory			.51
Anterior circulation	31 (60.8%)	12 (70.6%)	
Posterior circulation	12 (23.5%)	4 (23.5%)	
Ant and post	6 (11.8%)	0 (0%)	
Undetermined	2 (3.9%)	1 (5.9%)	
Baseline NIHSS	4 (2.9-12)	11.5 (2-16)	.37
Baseline ASPECTS > 5	43 (91.5%)	15 (88.2%)	1.00
Good baseline collaterals	10 (71.4%)	5 (55.6%)	.74
Acute stroke treatment			.26
No acute treatment	37 (72.5%)	9 (52.9%)	
IV-rtPA	12 (23.5%)H	6 (35.3%)	
Bridging	2 (3.9%)	2 (11.8%)	
Haemorragic transformation	2 (5%)	4 (25%)	.09
Stroke etiology (TOAST)			.00
LAA	4 (7.8%)	1 (5.9%)	
CE	18 (35.3%)	2 (11.8%)	
SVD	11 (21.6%)	0 (0%)	
Dissection	6 (11.8%)	11 (64.7%)	
Multiple causes	1 (2%)	0 (0%)	
Undetermined etiology	11 (21.6%)	3 (17.6%)	
Discharge therapy			.13
No discharge therapy	1 (2%)	0 (0%)	
Antiplatelets	39 (76.5%)	10 (58.8%)	
Anticoagulants	8 (15.7%)	7 (41.2%)	
Antiplatelets plus anticoagulants	3 (5.9%)	0 (0%)	
3-months mRS 0-2	41 (83.7%)	14 (82.3%)	1.00
3-months mortality	1 (2%)	0 (0%)	1.00
1 year stroke recurrence	5 (10.4%)	1 (5.9%)	.95

Abbreviations: CE, cardioembolic; LLA, large artery atherosclerosis; SVD, small vessels disease; NIHSS, National Institute of Health Stroke Scale; TOAST, Trial of Org 10172 in Acute Stroke Treatment.

Table 2. *Descriptive features of patients with ski-related stroke*

Age	Sex	TOAST	Risk factors list	Trauma description	Preceding symptoms during ski	Stroke onset timing	Delay	Clinical presentation	NIHSS	Arteries dissected	Interv type	mRS 3 m
54	M	D	AH; HL; Smoke	Minor ski fall	CBH	During ski, 90 min after fall	0	Paresis	1	Unilat. VA	None	3
42	M	D	AH; HL; Smoke	Ski fall with neck hyperestension	Cervical pain after ski	Days after	10	Paresis; Sensory deifict; Dysarthria; Neglect	11	Unilat. VA	IV rtPA	1
51	M	D	HL; Migraine	No trauma or fall	None	Morning after ski	1	Vigilance alteration; Visual field defect; Paresis; Sensory deifict; Dysarthria	16	Bilat. ICA	EVT	4
51	M	D	HL	Minor ski fall	None	During ski, 30 min after fall	0	Visual field defect; Paresis; Sensory deifict; Dysarthria; Neglect	16	Unilat. ICA	IV rtPA	2
47	M	D	HL; Smoke	Fall without loss of consciousness	Back and neck pain after the fall	Days after	15	Visual field defect; Paresis; Sensory deifict; Dysarthria; Neglect	15	Unilat. ICA	None	3
52	F	D	Smoke	No trauma or fall	None	Same day, after ski	0	Visual field defect; Paresis; Sensory deifict; Dysarthria; Neglect	18	Unilat. ICA	IV rtPA	0
24	F	D	Smoke	Low speed fall on the back, not cervical or head trauma	TIA immediately after fall, while standing up	Next day	1	Aphasia; Dysarthria	2	Unilat. ICA	None	0
35	M	D	Smoke	Major fall (3m high), head trauma wearing helmet, no loss of consciousness	None	Days after	2	Vigilance alteration; Paresis; Dysarthria	10	Unilat. VA	None	2
47	M	D	HL	Fall without loss of consciousness	Cervical pain after ski	Days after	31	Paresis; Sensory deifict; Dysarthria; Neglect	12	Bilat. ICA	IV rtPA	2
39	F	D		Repeaed falls, no loss of consciousness, TIA on day -4 and -1	First TIA same day of fall	Days after	8	Vigilance alteration; Paresis;	19	Unilat. ICA	IV rtPA	0

(*Continued*)

Table 2. (*Continued*)

Age	Sex	TOAST	Risk factors list	Trauma description	Preceding symptoms during ski	Stroke onset timing	Delay	Clinical presentation	NIHSS	Arteries dissected	Interv type	mRS 3 m
								Sensory deificit; Aphasia				
48	M	D	Migraine; PFO	Low speed ski fall, not cervical or cranic traumatism	None	Days after	5	Vigilance alteration; Eye deviation; Visual field defect; Paresis; Sensory deificit; Aphasia; Neglect	NA	Unilat. ICA	None	2
54	M	LAA	AH; HL	No trauma or fall	None	During ski	0	Visual field defect; Paresis; Sensory deificit; Dysarthria; Neglect	18		IV rtPA	1
62	M	UND	AH; HL	No trauma or fall	None	During ski (during downhill)	0	Dysarthria	2		None	1
66	M	UND	AH; Diabetes; HL; Alcohol abusel	No trauma or fall	None	During ski (break)	0	Paresis; Dysarthria	3		None	1
71	M	CE	HL; AF	No trauma or fall	None	During ski (start of day)	0	Visual field defect; Paresis; Sensory deificit; Aphasia; Dysarthria	15		EVT	2
51	M	CE	HL; AF	No trauma or fall	None	During ski (teleski)	0	Monocular blindness	0		None	0
54	F	UND	AH; HL	No trauma or fall	None	Same day, after ski	0	Paresis; Dysarthria	2		None	0

Abbreviations: AF, atrial fibrillation; AH, arterial hypertension; CBH, Claude-Bernard-Horner syndrome; CE, cardioembolic; D, dissection; EVT, endovascular treatment; F, female; HL, hyperlipidemia; ICA, internal carotid artery; LLA, large artery atherosclerosis; M, male; UND, undetermined; TOAST, Trial of Org 10172 in Acute Stroke Treatment; VA, vertebral artery.

Table 3. *Comparison between skiing-related and nonskiing-related dissections in ASTRAL registry*

	Dissections non ski-associated (n = 147)	Ski-associated dissections (n = 11)	*P* value
Age	47.9 (40.7-54.3)	46.9 (39.3-51.2)	.51
Female sex	67 (45.6%)	3 (27.3%)	.39
mRS prestroke	0 (0-0)	0 (0-0)	.94
Previous stroke	7 (4.8%)	0 (0%)	1.00
Hypertension	43 (29.2%)	2 (18.2%)	.66
Diabetes	5 (3.4%)	0 (0%)	1.00
Hypercholesterolemia	69 (46.9%)	6 (54.5%)	.86
Smoking	35 (23.8%)	6 (54.5%)	.06
Atrial fibrillation	0 (0%)	0 (0%)	1.00
Coronary artery disease	2 (1.4%)	0 (0%)	1.00
Patent forame ovale	8 (19.1%)	1 (16.7%)	1.00
Active cancer	2 (1.4%)	0 (0%)	1.00
Body Mass Index	25 (22.7-28)	26 (23-26.8)	1.00
Recent trauma			.00
No	118 (80.3%)	2 (18.2%)	
Minor	23 (15.7%)	8 (72.7%)	
Major	6 (4.1%)	1 (9.1%)	
Antiplatelets	9 (6.1%)	0 (0%)	.86
Anticoagulants	4 (2.7%)	0 (0%)	1.00
Antihypertensives	21 (14.3%)	1 (9.1%)	.98
Statins	15 (10.2%)	0 (0%)	.56
Stroke arterial territory			.93
Anterior circulation	106 (72.1%)	8 (72.7%)	
Posterior circulation	39 (26.5%)	3 (27.3%)	
Undetermined	2 (1.4%)	0 (0%)	
Baseline NIHSS	10 (4-18)	13.5 (9.3-16.2)	.70
Baseline ASPECTS >5	112 (79.4%)	9 (81.8%)	1.00
Good baseline collaterals	44 (56.4%)	3 (42.9%)	.77
Arteries dissected			.20
Single ICA	97 (66%)	6 (54.5%)	
Single VA	35 (23.8%)	3 (27.3%)	
Bilateral ICA	5 (3.4%)	2 (18.2%)	
Bilateral VA	5 (3.4%)	0 (0%)	
ICA and VA	5 (3.4%)	0 (0%)	
Grade of stenosis			.05
No stenosis	5 (3.4%)	2 (18.2%)	
Stenosis <50%	19 (12.9%)	3 (27.3%)	
Stenosis 50%-99%	33 (22.4%)	1 (9.1%)	
Occlusion	90 (61.2%)	5 (45.5%)	
Acute stroke treatment			.60
No acute treatment	87 (59.2%)	5 (45.5%)	
IV-rtPA	39 (26.5%)	5 (45.5%)	
Bridging	20 (13.6%)	1 (9.1%)	
MT only	1 (.7%)	0 (0%)	
Haemorragic transformation	25 (18.8%)	2 (18.2%)	1.00
Stroke etiology (TOAST)	0 (0%)	0 (0%)	1.00
Discharge therapy			.43
No discharge therapy	16 (10.9%)	0 (0%)	
Antiplatelets	90 (61.2%)	6 (54.5%)	
Anticoagulants	39 (26.5%)	5 (45.5%)	
Antiplatelets plus anticoagulants	2 (1.4%)	0 (0%)	
3-months mRS 0-2	83 (58.9%)	8 (72.7%)	.56
3-months mortality	13 (9.2%)	0 (0%)	.62
1 year stroke recurrence	16 (11.2%)	0 (0%)	.51

Abbreviations: ICA, internal carotid artery; Trial of Org 10172 in Acute Stroke Treatment; VA, vertebral artery.

preceding trauma in these patients was significantly higher than in stroke caused by nonskiing-associated dissection.

A previous case series of 12 patients already suggested a relationship between skiing and cervical artery dissections that our results seem to confirm. None of our patients had clinical or radiological evidence of collagen pathology or fibromuscular dysplasia and only 1 reported a severe trauma during skiing. Patients with arterial dissections might present an underlying arteriopathy, either genetically determined or related to transient environmental factors, such as infections.[8,9] In cases of arterial fragility, minor traumatism from falls during skiing may be a trigger to induce an arterial dissection[2]; given that 2 of our patients with SAS dissections had no trauma, acceleration forces, or nonviolent neck movements during downhill skiing may represent another underlying mechanism.

Previous European data suggest a higher prevalence of stroke from dissections in men.[9,10] This effect was even more pronounced in our cohort of ski-associated dissections, possibly because men are more prone to traumatism during skiing.[4] Similar to skiing, other activities have been related to cervical artery dissection, such as neck manipulation, giving birth, and various sports, where dissections may be secondary to direct cervical trauma (chiropractic manipulation, martial arts), or indirect mechanical solicitation of neck structures.[9,11,12]

For the one third of our SASs with etiologies other than dissection, the temporal association with ski practice may have been incidental. Alternatively, sustained physical effort at higher altitudes with dehydration and hemoconcentration, sympathetic activation and activation of prothrombotic factors may be implicated.[13-16]

Strengths of our study are the long observation period, the prespecified collection of data, and a large comparison sample matched for age and gender derived from the same population. Limitations are the retrospective, nonrandomized analysis of the study, the small sample size, the possible recall bias for mechanism of trauma in SAS, the absence of detailed recording of traumatic factors in the non-SAS dissection controls, and the single-center nature of the case series limiting generalizability. Finally, we might have underestimated the prevalence of associated collagen disease, since genetic evaluation was not systematically performed in our patients.

In conclusion, our results confirm the relationship between ski-practice and stroke from arterial dissection, even in the absence of severe traumatism. Occasionally ski-associated stroke might be secondary to other etiologies such as cardiac or arterial embolism.

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